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NOV 17 2004

FACSIMILE TRANSMISSION**DATE:** November 17, 2004**MATTER NUMBER:** 01030 10109097

RECIPIENT(s):	FAX No.:	PHONE No.:
Examiner Fozia M. Hamud Art Unit 1647 Commissioner for Patents	1571-273-0884 1703-872-9306 ✓	1571-272-0884

FROM: Norman Hanson**FLOOR:** 24**PHONE:** (212) 318-3168**FAX:** (212) 318-3400

U.S. Serial No. 10/026, 106

RE: LUD 5752**NUMBER OF PAGES INCLUDING COVER PAGE:****Message:****CAUTION - CONFIDENTIAL**

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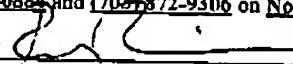
NOV 17 2004

LUD 5752 (10109097)

CERTIFICATE OF FACSIMILE TRANSMITTAL

I hereby certify that this correspondence is being transmitted via FACSIMILE pursuant to 37 CFR 1.8 to Group 1647, Examiner Fozia M. Hamud of the Commissioner for Patent at (571) 273-0884 and (703) 872-9306 on Nov. 17, 2004.

Sheila Murtha
(Name of Transmitter)


(Signature)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application of: Jean-Christophe RENAUD et al.

US Serial No.: 10/026,106

Group Art Unit: 1647

Filing Date: December 21, 2001

Examiner: Fozia M. HAMUD

For: ISOLATED CYTOKINE RECEPTOR LICR-2

LETTER
SUBMISSION OF DECLARATION
(37 C.F.R. § 1.116)

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dear Sir:

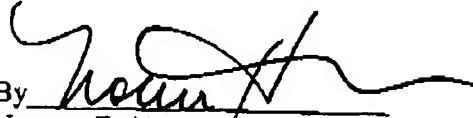
In support of the patentability of this invention, applicants submit the accompanying declarations.

There are two declarations, but they are copies of the same declaration. The inventors are at different locations, hence the need for two.

LUD 5752 (10109097)

It is requested that this material be considered in this case.

Respectfully submitted,

By 

Norman D. Hanson

Registration No.: 30,946

FULBRIGHT & JAWORSKI L.L.P.

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New York, New York 10103

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Attorneys for Applicant

Attachment: Letter Submission of Declaration

Received 11/16/2004 05:13 in 02:04 on line [3] for NH01030 Printed 11/16/2004 07:26 * Pg 2/7
 16 NOV 2004 10:15:22 LUDWIG INSTITUTE FULBRIGHT JAWORSKI NO. 53054 P. 24 03/05
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NOV 17 2004

LUD 5752 (10109097)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Applicant : Jean-Christophe RENAUD et al.
 Serial No. : 10/026,106
 Filed : December 21, 2001
 For : ISOLATED CYTOKINE RECEPTOR LICR-2
 Art Unit : 1647
 Examiner : Fozia M. HAMUD

Commissioner for Patents
 P.O. Box 1450
 Alexandria, VA 22313-1450

DECLARATION

The undersigned hereby declare as follows:

1. We are the correctly named inventors of the above referenced application, and are fully familiar with it.
2. We wish to elaborate on the experiments described in example 7 of this application, i.e., the ability of LICR-2 to activate STAT.
3. The cell line used in example 7 is known to express most of the STAT molecules.
4. Interleukin-29, or "IL-29", or "IFN2-1" is also known to be involved in the activation of STAT3. See WO 02/086087.
5. We carried out experiments in which antiserum against LICR-2 was made and used. cDNA for LICR-2, as described in the application, was cloned into a vector,

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16: NOV 2004 10:15:13 212 LUDWIG INSTITUTE FOR MEDICAL RESEARCH
LUDWIG INSTITUTE 3400 FULBRIGHT JAWORSKI NO. 53054 P. 34 04/25

LUD 5752 (1010097)

i.e., pEF-BOS-puro, using standard methods, and recombinant constructs were transfected into murine mastocytoma cell line P815. These cell lines were then transplanted via injection into syngenic DBA/2 mice. In a first injection, 300,000 cells were used, in a subcutaneous footpad injection, which was followed about 40 days later, with 400,000 cells, via the same mode. Two weeks later, a third injection, of 2 million cells, was administered intraperitoneally.

6. Sera were collected, 22 days after the first injection, and then just before the third injection. Standard assays established that the antisera were specific for LICR-2.
7. The antisera were then used, in luciferase assays as described in example 7 where BW5147 cells were transfected with wild type, LICR-2 receptor. The cells were stimulated with IL-29 (25 ng/ml) and luciferase production was upregulated by IL-29. In a parallel set of experiments, when the cells were preincubated with the LICR-2 antisera, no luciferase upregulation by IL-29 was seen.
8. The experiments in "7" were repeated, and the phosphorylation of STAT3 was measured, using standard assays. Phosphorylation of STAT3 is a prerequisite to its activation. No phosphorylation was observed in the presence of anti-LICR-2 serum.
9. These experiments lead to the conclusion that LICR-2 is in fact involved in STAT activation. STAT3 in particular.

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16 NOV 2004 10:15 21 LUDWIG INSTITUTE FOR CANCER RESEARCH FULBRIGHT&JAWORSKI NO. 53054 P. 44 85/85
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LUD 8782 (10109097)

10. We hereby declare that all statements made herein of our own knowledge are true, and that all statements made on information or belief are believed to be true; and further that these statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements and the like so made may jeopardize the validity of this declaration, the subject application, or any patent issued thereon.

November 3, 2004

Date



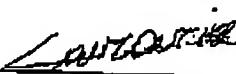
Jean-Christophe RENAULD

Date

Helmut FICKENSCHER

November 3, 2004

Date


Laure DUMOUTIER

November 9, 2004

Date


Simon HÖR

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NOV 17 2004

LUD 6762 (10109097)

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Applicant : Jean-Christophe RENAULD et al.
Serial No. : 10/026,106
Filed : December 21, 2001
For : ISOLATED CYTOKINE RECEPTOR LICR-2
Art Unit : 1647
Examiner : Farzla M. HAMUD

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

DECLARATION

The undersigned hereby declare as follows:

1. We are the correctly named inventors of the above referenced application, and are fully familiar with it.
2. We wish to elaborate on the experiments described in example 7 of this application, i.e., the ability of LICR-2 to activate STAT.
3. The cell line used in example 7 is known to express most of the STAT molecules.
4. Interleukin-29, or "IL-29", or "IFNA-1" is also known to be involved in the activation of STAT3. See WO 02/086087.
5. We carried out experiments in which antiserum against LICR-2 was made and used. cDNA for LICR-2, as described in the application, was cloned into a vector,

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LUDWIG INSTITUTE 400

LUD 5752 (10109897)

i.e., pEF-BOS-puro, using standard methods, and recombinant constructs were transfected into murine mastocytoma cell line P815. These cell lines were then transplanted via injection into syngenic DBA/2 mice. In a first injection, 300,000 cells were used, in a subcutaneous footpad injection, which was followed about 40 days later, with 400,000 cells, via the same mode. Two weeks later, a third injection, of 2 million cells, was administered intraperitoneally.

6. Sera were collected, 22 days after the first injection, and then just before the third injection. Standard assays established that the antisera were specific for LICR-2.
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8. The experiments in "7" were repeated, and the phosphorylation of STAT3 was measured, using standard assays. Phosphorylation of STAT3 is a prerequisite to its activation. No phosphorylation was observed in the presence of anti-LICR-2 serum.
9. These experiments lead to the conclusion that LICR-2 is in fact involved in STAT activation, STAT3 in particular.

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LUDWIG INSTITUTE 3400

LUD 5752 (10109097)

10. We hereby declare that all statements made herein of our own knowledge are true, and that all statements made on information or belief are believed to be true; and further that these statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements and the like so made may jeopardize the validity of this declaration, the subject application, or any patent issued thereon.

November 3, 2004

Date

Jean-Christophe RENAULT
Jean-Christophe RENAULT

November 9, 2004

Date

Helmut Fickenscher
Helmut FICKENSCHER

November 3, 2004

Date

Laure Dumoutier
Laure DUMOUTIER

Date

Simon Hör
Simon HÖR

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